Medical Progress

Abdominal Aortic Aneurysms

GEORGE FORTNER, MD, *and* KAJ JOHANSEN, MD, PhD, *Seattle*

Aneurysms are common in our increasingly elderly population, and are a major threat to life and limb. Until the advent of vascular reconstructive techniques, aneurysm patients were subject to an overwhelming risk of death from exsanguination. The first successful repair of an abdominal aortic aneurysm using an interposed arterial homograft was reported by Dubost in 1952. A milestone in the evolution of vascular surgery, this event and subsequent diagnostic, operative and prosthetic graft refinements have permitted patients with an unruptured abdominal aortic aneurysm to enjoy a better prognosis than patients with almost any other form of major systemic illness.

(Fortner G, Johansen K: Abdominal aortic aneurysms [Medical Progress]. West J Med 1984 Jan; 140:50-59.)

An aneurysm is a balloonlike dilatation of an artery that frequently presents as a pulsatile mass. Such lesions have long been recognized, classically in the extremities, as a result of infection or trauma. In the second century AD Antyllus gave a clear description of aneurysms, including a taxonomy related to such lesions' potential for rupture. Galen described aneurysms due to trauma, especially from an unskillful phlebotomy, and noted that rupture could be forestalled in some circumstances by external compression. In the fifth century AD Aëtius clearly described a now well-established principle of operative treatment of aneurysms in which proximal arterial control is followed by ligature from within the aneurysm of the orifices of inflow and outflow vessels. Vesalius offered the first clinical description of an aneurysm of the abdominal aorta, and his 16th-century contemporary Ambroise Paré pointed out that arterial wall degeneration can be caused by syphilis, that aneurysms may thrombose and that injudicious incision of an inflamed pulsatile mass may cause exsanguinating hemorrhage. Morgagni observed yet another complication of aneurysms—erosion of the vertebral bodies by a subjacent aortic aneurysm. William and John Hunter, who made significant advances in both the physiology and surgical repair of blood vessels, pioneered in the treatment of peripheral aneurysms, and John Hunter's work on collateral blood flow led to a much easier approach to these lesions—proximal arterial occlusion by ligature. One of Hunter's pupils, Sir Astley Cooper, used the Hunterian approach for common carotid and internal and external iliac artery aneurysms and in 1817 ligated the abdominal aorta proximal to a leaking left iliac artery aneurysm.

Aneurysms tend to enlarge, and their risk of rupture rises geometrically with their size. Numerous novel attempts were made to halt the inexorable growth of aneurysms in the decades before the development of modern vascular surgical techniques. Teams of medical personnel were enlisted to provide external compression, sometimes for days at a time, in patients with large abdominal aneurysms. Arterial walls were scarified with talc, wrapped in cellophane or filled with many meters of wire to which a galvanic current might be applied. This latter technique—aneurysmal wiring and electrocoagulation—was reported favorably as late as 1951 by Blakemore.[1] Matas of New Orleans renovated the approach of Aëtius and carried out endoaneurysmal ligature of peripheral aneurysms: such an approach is important because it spares collaterals around the aneurysm. Creech, a pupil of Matas, was later able to point out the virtues of the intrasaccular graft interposition now universally used for aortic aneurysmal repair.[2]

Interestingly, whereas peripheral aneurysms due to trauma or bacterial infection were common in the past, aneurysms of the abdominal aorta appear to be a modern disease. Osler reported seeing only one such lesion per year, similar to the incidence noted at Guy's Hospital in London in the mid-19th century.[3]

From the Department of Surgery, University of Washington School of Medicine, Seattle.
Reprint requests to Kaj Johansen, MD, PhD, Department of Surgery, Harborview Medical Center, 325 Ninth Avenue, Seattle, WA 98104.

Epidemiology

Osler would be surprised today: abdominal aortic aneurysm can be found in at least 2% of the elderly population, according to a Scandinavian postmortem study,[4] and 10% or more of the average vascular surgeon's clinical practice is comprised of patients who have abdominal aortic aneurysms.[5] Current demographic trends in Western nations, showing an ever-increasing elderly population, will undoubtedly account for an even greater increase in the number of patients with this condition.

Men outnumber women by fivefold to tenfold in susceptibility to abdominal aortic aneurysm. The vast majority of persons with abdominal aortic aneurysm are older than 60 years of age, and the median age of persons so afflicted in several large series was 70.[6] No convincing racial, environmental or geographic predilection has been noted.

Etiology

For such a common condition, surprisingly little is understood about the cause of aneurysms. They are frequently thought to be a manifestation of atherosclerosis, perhaps because of the endemic nature of this latter condition and the not infrequent concurrence of the two. It is clear, however, that aneurysms arise primarily because of deterioration in the arterial *media*. Far-advanced atherosclerosis may have both direct and indirect effects upon this layer of the artery, but it is primarily an *intimal* disease. The presentation and clinical consequences of aortic atherosclerosis are very different from those of aneurysmal disease. It has been suggested that abdominal aortic aneurysms may develop as the result of a series of structural, biochemical and hydraulic factors independent from those usually associated with the development of atherosclerosis.

With increasing age, aortic elasticity and compliance diminish, and increasing wall rigidity can be shown experimentally to cause development of a reflected wave from the aortic bifurcation. Gosling and colleagues[7] have suggested that this retrograde pressure wave causes a lateral stress on the infrarenal abdominal aorta, especially when it meets the next oncoming pulse wave. Further, Roach[8] found that turbulent flow generated distal to the branching of large tributaries, such as the renal arteries, may initiate vibrations in the arterial wall that themselves may cause medial degeneration. With deterioration of the wall's elastic lamellae, arterial wall tensile strength declines and the artery dilates in response to the intraluminal pressure, usually greater than 2.5 lbs per sq in (17.25 kPa). The aorta dilates in concert with Laplace's law ($T = P \times r$), where wall tension (T) is a function of the product of intraluminal pressure (P) and aortic radius (r). (Parenthetically, Laplace's law clarifies the role of sustained hypertension in the genesis of aneurysms.) That such hydraulic factors may play a role in the development of aneurysms seems supported by the fact that aneurysms develop throughout the body in segments of large and medium-sized arteries just proximal to bifurcations or branch points.

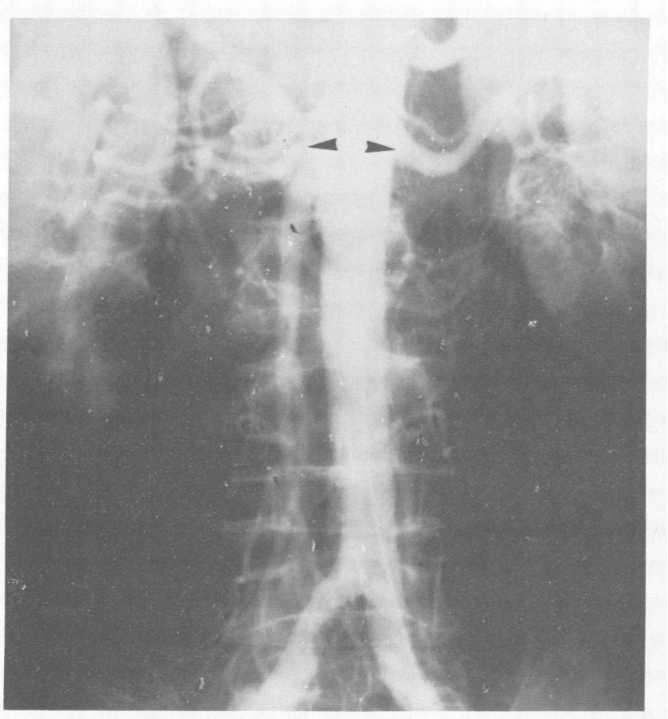

Figure 1.—A normal abdominal aortogram. Aneurysms characteristically arise between renal arteries **(arrows)** and the aortic bifurcation.

The size, shape and structure of the aorta may be germane to the development of aneurysmal degeneration. Comparative anatomic studies have shown that the human abdominal aorta may be structurally deficient in both its complement of vasa vasorum (the small arteries supplying nutrition to the outer aspects of the aortic wall) and in the number of musculoelastic units found in the media.[9] While the normal human aorta has the configuration noted in Figure 1, it frequently "uncoils" with age, becoming extremely tortuous ("ectatic"), a common finding in aneurysmal aortas (Figure 2). "Uncoiling" of the aorta may weaken its structural elements. Tilson and Dang[10] have observed that the arteries of persons with aneurysms are increased in diameter when compared with those of the normal population—a condition they call "arteriomegaly." It has also been observed that aneurysms generally arise in segments of arteries that cross joints, giving off few branches—for example, the infrarenal aorta and the popliteal artery. Repeated flexing of the artery could conceivably cause structural weakening over many decades. Thus, such uncoiled, dilated, structurally deficient and stressed segments of aorta might seem to be especially susceptible to further weakening and ballooning.

Several biochemical factors may further contribute to the pathogenesis of aneurysms. Aortic aneurysms have been shown to develop spontaneously in laboratory mice with the "blotchy" allele on the X chromosome.[11] This mutation is expressed by inhibition of

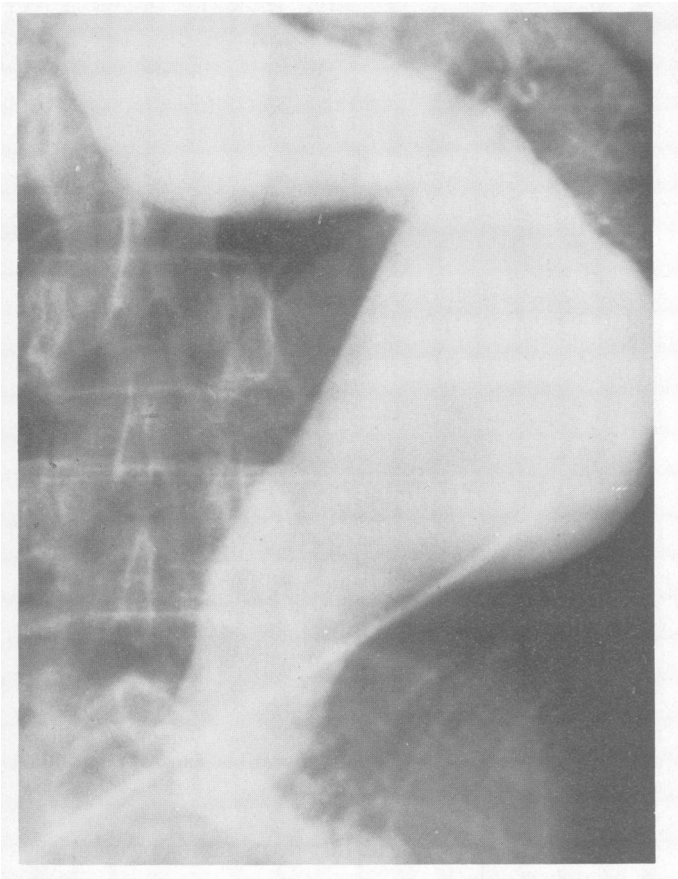

Figure 2.—Marked ectasia ("uncoiling") of the abdominal aorta, with an associated large aortic aneurysm.

the collagen-elastin bridging mediated by lysyl oxidase, a copper-containing enzyme. Interestingly, Menkes's kinky-hair syndrome, a rare sex-linked condition in newborn human infants characterized by collagen abnormalities and malabsorption of copper, may also be associated with arterial aneurysms.[12] Busuttil and co-workers[13] and Dobrin[14] have suggested that dissolution of the structural integrity of the aortic wall by collagenase may lead to aortic dilatation and subsequent rupture of aortic aneurysms. Collagenase levels are significantly higher in the walls of ruptured aneurysms when compared with unruptured abdominal aortic aneurysms or atherosclerotic aortas.[13] Adding further clinical significance to these experimental studies has been the observation[15] that patients with aortic aneurysms who undergo laparotomy (which liberates large amounts of collagenase) for other purposes may be at increased risk postoperatively for aneurysmal rupture. The possibility that a chromosomal defect, expressed as an enzymatic abnormality, leads to arterial wall deterioration lends credibility to a possible inherited tendency to form aneurysms. We are currently engaged in a population study to attempt to determine whether relatives of aneurysm patients are at excess risk of aneurysm development as well.

In short, the presence of multiple risk factors independent of those associated with atherosclerosis, as well as the relative rarity of aneurysmal degeneration in aortas severely involved with occlusive atherosclerosis, suggests that aneurysmal degeneration and atherosclerosis may be independent processes.[16]

Rare in modern experience, but worthy of mention, are the occasional aortic aneurysms that arise due to tuberculosis,[17] syphilis[18] and other bacterial infections.[19] Abdominal aortic aneurysm is also a manifestation of connective tissue disorders such as Ehlers-Danlos[18] or Marfan's[20] syndromes or pseudoxanthoma elasticum.[18] Aneurysm may occasionally develop as a consequence of aortic dissection[21] or blunt or penetrating trauma.[22]

Pathology

A normal adult infrarenal aorta measures 1.8 cm in diameter. What degree of aortic dilatation constitutes an abdominal aortic aneurysm? The literature is unclear on this question, but most would agree that an aorta is "aneurysmal" when the aortic diameter doubles —that is, exceeds 3.5 cm. In all, 95% of abdominal aortic aneurysms originate distal to the renal artery orifices and terminate proximal to the aortic bifurcation. Typically the aneurysm is a fusiform dilatation of the vessel that involves the full thickness of the wall, thereby being a "true" aneurysm. "False" aneurysms—those involving less than the three arterial layers—and aortic dissections are uncommon in this area. Turbulent blood flow within the aneurysm results in the deposition of a thick layer of fibrin, platelet aggregates and entrapped erythrocytes along the intimal surface—a so-called mural thrombus. Vessels originating from the aneurysmal portion of the aorta, such as the inferior mesenteric and lumbar arteries, may be obliterated by this coagulum. Further, the mural thrombus may fragment and ulcerate, embolizing the distal vessels, or it may thicken and occlude the aortic lumen, causing complete interruption of flow through the aorta.

The aneurysmal aortic wall exhibits progressive degeneration of the normal arterial architecture, with disruption of endothelium and necrosis of the smooth muscle cells. Calcification of the aneurysmal wall occurs frequently. The adventitia usually becomes attenuated and may fuse with nearby vital structures such as the inferior vena cava, the ureters or the gastrointestinal tract. The aortic wall may rarely thicken and become infiltrated with leukocytes, round cells and collagen, forming an "inflammatory" aneurysm, at which time the entire retroperitoneum becomes an impenetrable adherent mass.

Clinical Manifestations

Most abdominal aortic aneurysms are asymptomatic. Thus, in most circumstances they are discovered during the course of routine physical examination or diagnostic studies done for other reasons. Because the aortic bifurcation corresponds roughly with the umbilicus, abdominal aortic aneurysms are usually palpated in the epigastrium. The wall calcifications of an aneurysm may betray its presence on plain films of the abdomen obtained for other purposes. In recent years small aortic aneurysms have been more frequently noted as

an incidental finding during noninvasive imaging in patients having these studies for the evaluation of other problems.

Abdominal aortic aneurysms may be associated with diverse symptoms, depending on how the aneurysm deranges a patient's anatomy and physiology. Simple expansion of the aneurysm can compress and erode neighboring structures (duodenum, stomach, vena cava, ureter or vertebral bodies) and produce symptoms referrable to these organ systems as they are insulted. As noted, a mural thrombus is capable of embolizing distal blood vessels. Rarely, the aneurysm can itself thrombose, causing catastrophic lower body ischemia.[23] Abdominal aortic aneurysms are susceptible to infection and can present as a source of occult sepsis.[17] Chronic consumptive coagulopathy has been reported.[24]

The most dramatic and catastrophic manifestation of abdominal aortic aneurysm occurs when the attenuated arterial wall yields at last to the intraluminal pressure and the aneurysm ruptures. This is a surgical emergency over which few others can take precedence. Frequently the ruptured aorta bleeds into the retroperitoneum, and for a time the hemorrhage is tamponaded. Patients may thus have a "leaking" abdominal aortic aneurysm in which back or flank pain is the cardinal symptom. This generally progresses rapidly to the exsanguinating hemorrhage of true rupture. If the aneurysm ruptures directly into the peritoneal cavity, as it may in up to 20% of cases, the patient rapidly dies from hypovolemic shock.[25]

The diagnostic triad associated with ruptured abdominal aortic aneurysm includes back, flank or abdominal pain, the presence of a pulsatile abdominal mass and shock. Such patients should be considered to have a ruptured aneurysm until proved otherwise by laparotomy; they should be taken to the operating suite without further delay due to diagnostic studies, placement of invasive monitoring devices or attempts at resuscitation. Prolonged hypoperfusion and tissue ischemia in these elderly patients is poorly tolerated and contributes to the further complications of renal failure, myocardial infarction, stroke and intestinal ischemia.[26] The management of a patient with a ruptured abdominal aortic aneurysm is discussed in greater detail later.

A pulsatile epigastric mass is not always an abdominal aortic aneurysm: a normal-sized but ectatic aorta may be confused with an aneurysm and occasionally such a finding will turn out to be normal aortic pulsations transmitted through a large overlying neoplastic or inflammatory mass.[27] The ability to palpate a free space between the costal margin and the convexity of the aneurysm suggests limitation of the aneurysm to the infrarenal aorta, a finding first noted by DeBakey.[28]

About 10% of these patients will have associated symptoms or signs of concomitant lower extremity occlusive disease—that is, complaints of claudication or rest pain, bruits, absence of distal pulses or an abnormal ankle-arm pressure index.[29]

Just as occlusive atherosclerosis may be silently present at multiple locations throughout the arterial tree, aneurysms may also be multifocal: 4% of patients with abdominal aortic aneurysms will have an aneurysm elsewhere, and those with peripheral arterial aneurysms, such as in the popliteal or femoral arteries, have a greater than 60% probability of having a concomitant abdominal aortic aneurysm.[30]

It is important to recall that these elderly patients have a high incidence of associated chronic illness, including coronary artery disease in up to 50% and hypertension in 25%.[29,31] These conditions, as well as other afflictions of the cardiovascular system, should be aggressively sought, as their appropriate management enhances the ultimate care of these patients.

Diagnostic Studies

Investigations that may be of value in a patient with an abdominal aortic aneurysm include standard anteroposterior and lateral abdominal x-ray films, abdominal ultrasonography, computed tomography (CT) and contrast arteriography. These studies are rarely indicated in a situation wherein ruptured aneurysm is

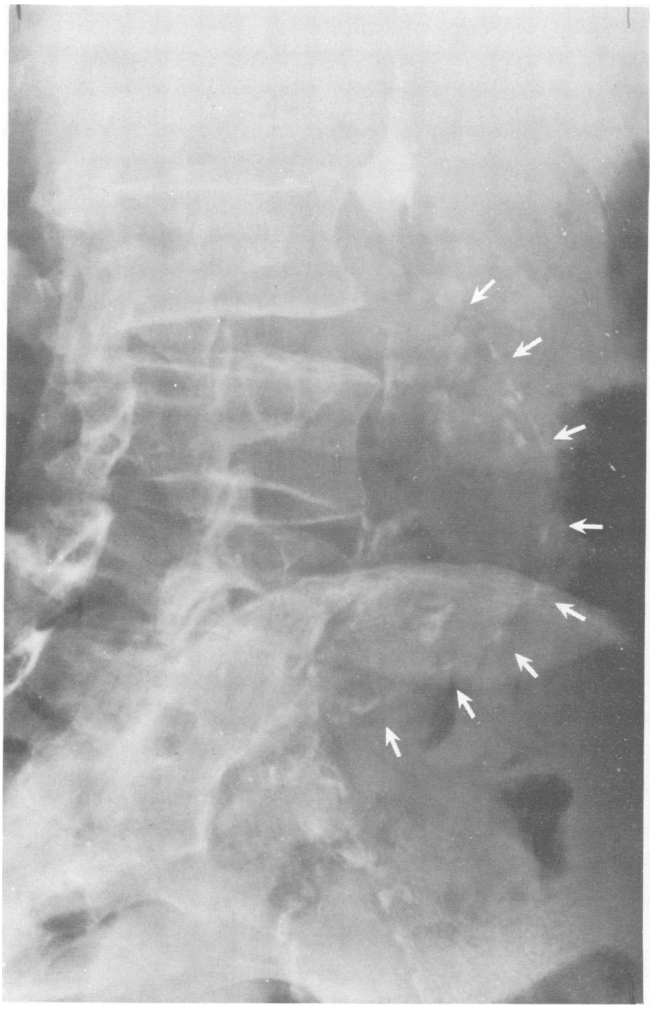

Figure 3.—Lateral abdominal roentgenogram showing calcification in the wall of an aortic aneurysm **(arrows).**

a consideration, but for patients with less worrisome manifestations of aneurysmal disease, selective use allows more appropriate treatment.

The calcified media of an aortic aneurysm is visible in up to 85% of such patients on plain abdominal films (Figure 3).[32] It should be recalled that the magnification effect of standard radiographic techniques tends to overestimate aneurysmal size, such that an aortic aneurysm measuring 6 cm on abdominal x-ray film will actually measure 5 cm at operation.[33]

Contrast angiography has become a relatively safe procedure for the evaluation of patients with vascular diseases. By outlining the arterial lumen with a column of radiopaque dye, one can determine the location of vital arterial branches originating from the aorta and determine the presence of any occlusive disease. The angiogram may show an aortocaval fistula or an anomalous renal blood supply. Many surgeons believe that angiography is routinely indicated for all patients who have abdominal aortic aneurysm,[34] whereas others reserve this procedure for those in whom there is clinical suspicion of involvement of the renal arteries, symptoms or noninvasive studies suggesting lower extremity occlusive disease, or a question of compromise of the mesenteric circulation.[35] The major limitations of angiography as a screening test for abdominal aortic aneurysm are its invasive nature and the fact that a mural thrombus may fill the lumen of an aneurysm to the extent that the aneurysmal contour does not visualize. Thus, contrast angiography is not useful for diagnosing the *presence* or *size* of an abdominal aortic aneurysm, but it is helpful in planning an operation in selected patients.

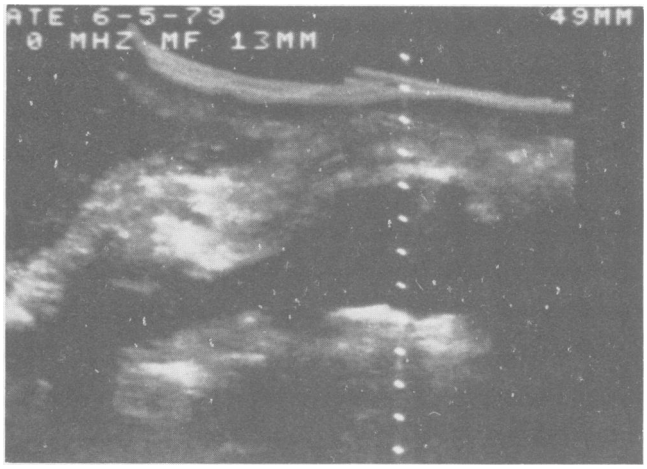

Figure 4.—B-mode abdominal ultrasonography both determines the presence of, and accurately measures, an aortic aneurysm.

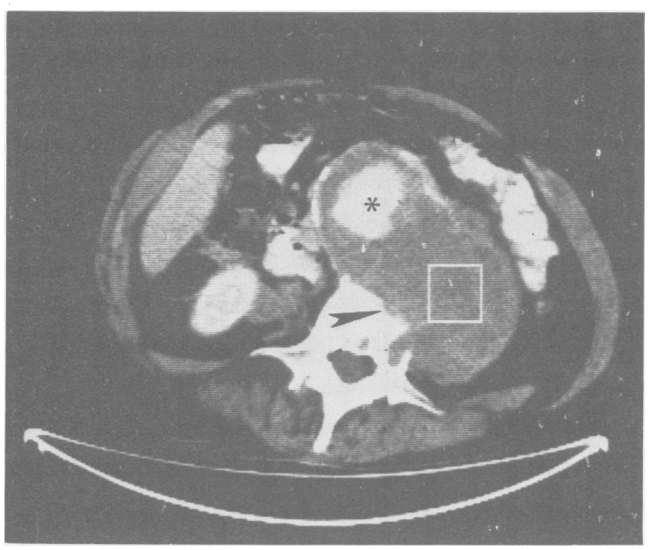

Figure 5.—Computed tomography defines a large, ruptured aortic aneurysm. Radiopaque contrast material has been administered, permitting delineation of the aortic lument (*) and its mural thrombus. Note erosion of the vertebral body by the aneurysm **(arrow).**

For routine demonstration and measurement of abdominal aneurysms, B-mode ultrasonography has the advantages of being noninvasive, painless, inexpensive, rapidly done and reproducible. It is easily done on an outpatient basis. Ultrasonography has nearly 100% sensitivity, the only difficulties being encountered in obese patients or in those with large amounts of intestinal gas.[36] B-mode ultrasonography also provides highly accurate measurements of aneurysmal dimension, essential in defining the indications for surgical intervention; it is also useful in providing serial measurements of aortic aneurysms over time in persons with equivocal indications for operation (Figure 4).[37] Finally, because aneurysms tend to occur in multiple locations, the ultrasound scan is also a good way to examine other areas in which aneurysmal degeneration is highly probable, such as the popliteal artery.[30]

Computed tomography consists of passing an x-ray source and detector array around a patient in a transverse plane, with computer-assisted generation of a cross-sectional image. The procedure may be done with intravascular contrast to provide precise definition of aortic anatomy. Multiple cross-sectional scans can delineate the aortic wall, mural thrombus and lumen for the entire length of the vessel (Figure 5).[38] Gas and fat do not interfere with the sensitivity of the study, and it is therefore useful in those patients not amenable to ultrasonography. In equivocal cases, CT is the best noninvasive method to search for evidence of aneurysmal rupture. Size estimates are highly accurate when compared with intraoperative findings.[32] The disadvantages of CT are its expense and the need for radiation exposure, both of which limit its use for screening and followup: also, CT may consume more time than is available to evaluate an unstable patient suspected of harboring a ruptured abdominal aortic aneurysm.

Review of the relative strengths and weaknesses of these diagnostic techniques suggests the following guidelines for their use:

- Patients with the classical clinical picture of ruptured abdominal aortic aneurysm require no further diagnostic studies.

- Patients who are relatively stable and in whom the diagnosis of ruptured abdominal aortic aneurysm is uncertain should be evaluated promptly by a CT scan.
- Stable persons who have abdominal aortic aneurysm in whom rupture is not a concern should be evaluated by ultrasonography to assess aortic dimensions. Many would recommend that this be followed by an angiogram if operation is planned and there is concern about suprarenal extension of the aneurysm, coincident arterial occlusive disease or the possibility of associated congenital anomalies such as horseshoe kidney, left-sided vena cava or atypical renal blood supply.

Natural History of an Untreated Asymptomatic Abdominal Aortic Aneurysm

Left unmanaged, abdominal aortic aneurysms usually enlarge, rupture and result in the death of the persons harboring them.[5,39,40] Once aneurysmal dilatation has started, immutable physical forces are in effect that, by Laplace's law, lead to progressive aneurysmal enlargement. What is the risk of rupture in a patient recently found to have abdominal aortic aneurysm?

In 1950, before the era of effective treatment of aortic aneurysms, Estes reported a study of 102 patients who had aortic aneurysms found on physical examination or abdominal x-ray film.[41] No comment was made regarding the dimensions of the aneurysms. Of the 102 patients, 60% survived for more than a year, but only 18.9% survived for five years. The survival of a synchronous age-matched population without abdominal aortic aneurysms was 80% at five years. In all, 63% of the aneurysm patients died of aneurysmal rupture. Klippel and Butcher reported similar statistics.[42]

The size of an aortic aneurysm is the single most important variable to consider in evaluating the risk of rupture. In 1955, Crane reported a 4% incidence of death due to rupture in patients with abdominal aortic aneurysms measuring less than 7 cm in diameter.[6] This finding has been confirmed in subsequent studies that have refined our appreciation of the relationship between aneurysmal size and risk of eventual rupture.[25] Contemporary thoughts about this relationship are summarized in the graph in Figure 6. We provide below our current indications for operative repair as they are affected by aneurysmal size.

Most aneurysms enlarge with time. Bernstein[43] used ultrasonography to follow a group of 49 high-risk patients with small (less than 6 cm in diameter) abdominal aortic aneurysms at three-month intervals. He reported an average growth rate of 0.4 cm per year, and proposed that a rate of growth in excess of this represents an increased risk of rupture.[37,43]

Indications for Operation

Prolongation of actuarial life expectancy, recognition of the natural history of untreated abdominal aortic

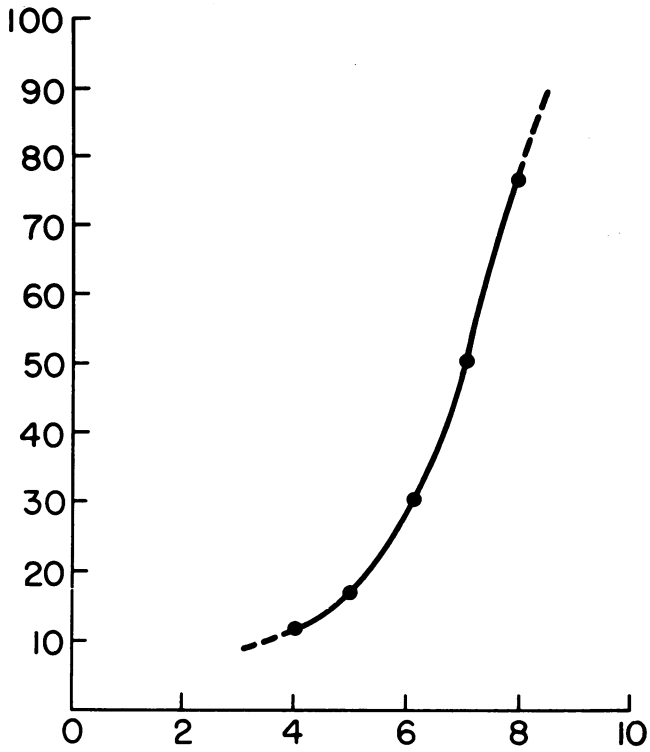

Figure 6.—Graph displaying chance of aneurysmal rupture within five years (Y axis, in percentage) as a function of diameter of aneurysm (X axis, in centimeters). (Redrawn from Rutherford.[5])

aneurysms and improving resuscitative, operative and intensive care skills have resulted in a liberalization of indications for operative treatment of these aneurysms.

Abdominal aortic aneurysm has become a major public health concern because life expectancy in the elderly now significantly exceeds the median age, 70, at which abdominal aortic aneurysms are diagnosed.[44] Untreated, rupture of an aortic aneurysm is lethal: even when managed in expert and timely fashion, mortality may exceed 50%[40] and may be greater than 80% if rupture is accompanied by shock.[45] A patient with aneurysmal rupture always requires urgent surgical intervention, except in those situations in which the presence of a widely disseminated neoplasm or other far-advanced and debilitating chronic illness renders operative therapy a disservice to the patient.

Other complications of abdominal aortic aneurysm —compression of neighboring structures, infection and embolization—also mandate surgical correction, but generally under less urgent circumstances, allowing adequate patient preparation and some prior knowledge of possible operative pitfalls. Similarly, aortic aneurysms that are tender or that have enlarged excessively on serial ultrasonograms generally warrant repair.

Recommendations for the repair of asymptomatic abdominal aortic aneurysms have classically been formulated on the basis of aneurysmal diameter, taking into account a patient's projected life span and the presence of associated medical conditions that might influence

operative risk. Very large abdominal aortic aneurysms, measuring 10 cm or more, are occasionally discovered on routine examination. Such lesions are at exceedingly high risk for rupture, and repair of the aneurysm should be considered urgent. Smaller aneurysms permit a more deliberate approach, but patients who have abdominal aortic aneurysms measuring 5 cm or greater by ultrasonogram or CT scan should undergo elective repair unless their operative risk is very high (recent myocardial infarction or stroke; far-advanced chronic illness). Patients who have aneurysms smaller than 5 cm in diameter should also be considered for operation if they are otherwise healthy: it deserves emphasis that small aortic aneurysms *do* rupture,[46] though less predictably than large ones. *All* abdominal aortic aneurysms are at some risk of rupture, and almost invariably the aneurysm is the patient's greatest threat to life; it is a physician's responsibility to minimize this risk.

Patients who have abdominal aneurysms smaller than 5 cm in diameter, and thus are marginal operative candidates, should be educated about premonitory symptoms of rapid expansion or imminent rupture. They should be closely followed and undergo serial abdominal ultrasonograms every three to six months to seek signs of rapid dilatation. If the aneurysm appears to be expanding at a rate greater than 0.4 cm per year, then the patient may be at a higher risk of rupture and should be considered a surgical candidate.[37,43]

Other factors prompting a more aggressive approach include inflammatory aneurysms, aneurysms that are not calcified on plain abdominal x-ray film (based on the argument that a noncalcified aneurysm has grown so rapidly that its wall has not had time to calcify) and aneurysms in persons who live far from tertiary surgical care. Elective aneurysm resection can be safely eschewed in an occasionally encountered patient whose aneurysm is small and who lives with other dependable people near skilled medical care, or a person whose life expectancy is short—less than six months—due to conditions such as metastatic malignancy or severe cardiopulmonary disease. Age is not a contraindication to operation: O'Donnell reported a 4.7% operative mortality in a series of octogenarians who underwent operation for abdominal aortic aneurysm.[47]

The outcome of operation for aneurysm has improved greatly because of major advances in preoperative evaluation, blood-banking, anesthesia and postoperative care.[48,49] The average mortality reported for elective aortic aneurysm repair should be 2% to 3% in most centers.[50,51] The outcome of aneurysm repair clearly depends on the surgeon's skill and experience, and the procedure should not be done by "occasional" vascular surgeons.[52]

Treatment

An explosion of vascular techniques resulted from the pioneering research of Gross, Crafoord, Lord and others in the 1930s and 1940s. Combined with burgeoning technologies in in vitro tissue preservation and the realization that cloth and, later, plastics could be formed into substitute vessels, these advances soon led to direct attack on abdominal aortic aneurysms. Oudot replaced the infrarenal abdominal aorta for occlusive disease in early 1951, and Dubost and colleagues followed soon thereafter with excision of an abdominal aortic aneurysm and restoration of arterial continuity with an arterial homograft. A ruptured

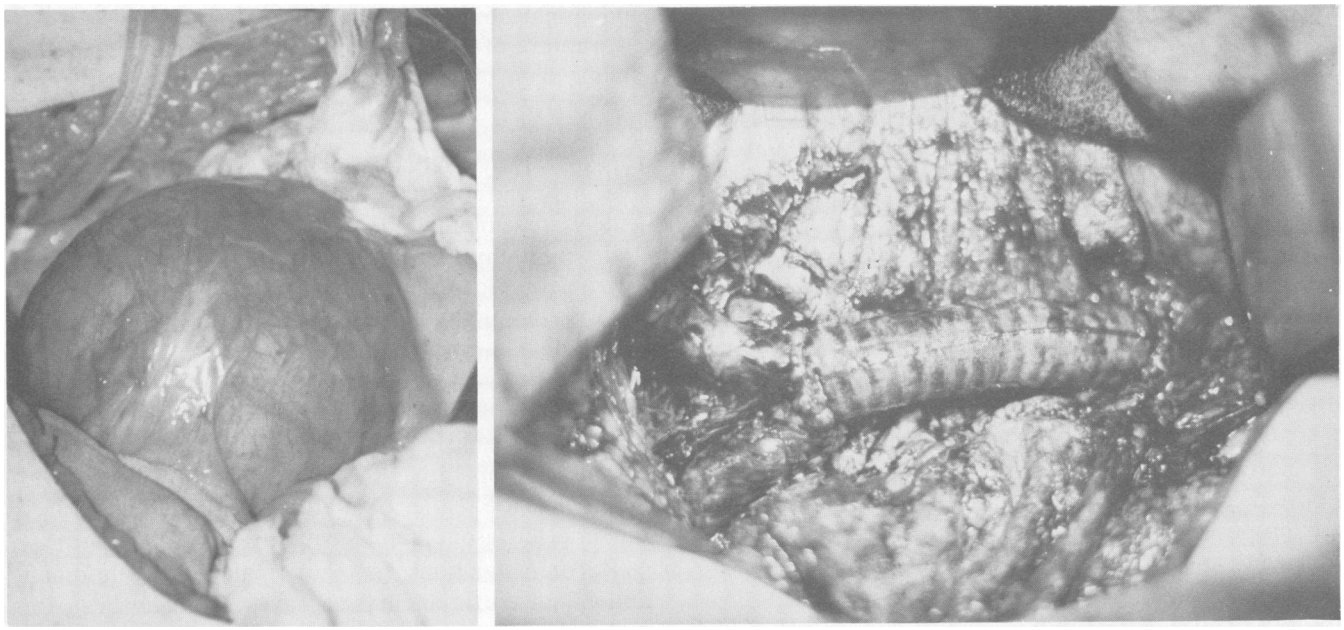

Figure 7.—Contemporary vascular surgical technique for a large aortic aneurysm included **(left)** control of proximal and distal vessels, opening the aneurysmal sac and **(right)** suturing a prosthetic graft of appropriate dimension to the inflow and outflow vessels from within.

aneurysm was controlled, excised and replaced with homograft by Cooley and DeBakey in 1953, and Rob and co-workers used a tube fashioned out of shirt linen to replace an abdominal aortic aneurysm in 1954. In 1966 Creech[2] pointed out that the excessive morbidity and mortality, primarily resulting from blood loss, associated with aneurysm excision could be avoided by an "endoaneurysmal" approach whereby, following proximal and distal arterial control, the aortic aneurysm is opened, the adherent mural thrombus evacuated and an appropriate graft sutured to the inflow and outflow vessels from within (Figure 7). This approach, a refinement of that suggested by Aëtius 1,400 years ago, remains to the present the standard operative approach to a "routine" infrarenal aortic aneurysm. When significant occlusive or aneurysmal disease is present in the iliac vessels, a bifurcation graft to the external iliac or femoral arteries is indicated.

Specific technical issues surrounding operation for an aneurysm are beyond the scope of this discussion. Important principles, however, include the assurance of adequate vascular access, the utility of preoperative volume loading and hemodynamic monitoring,[53] typing and crossmatching of adequate amounts of blood, the value of autotransfusion devices for the administration of shed autologous blood,[54] the crucial importance of carrying out the procedure rapidly and technically correctly, prevention of "declamping shock" (a characteristic hypotension following release of aortic occlusion)[55] and vigilance against the postoperative consequences of diverse types of tissue ischemia. Occasionally, debris embolized from the aorta during manipulation of an aneurysm may lodge distally, causing lower extremity ischemia or visceral infarction.[56]

The management of a patient with a ruptured abdominal aortic aneurysm frequently taxes a hospital and its physicians to the ultimate. While such patients may present initially with only back, flank or groin pain, this is frequently but a harbinger of complete vascular collapse. Even if repair of a ruptured abdominal aortic aneurysm is technically successful, the accompanying hypovolemic shock may result in stroke, myocardial infarction, kidney failure or colon gangrene, the latter condition associated with an 80% mortality in our experience.[45]

Whereas a "symptomatic" or "leaking" aneurysm may offer, in a technical sense, few differences from a routine elective abdominal aortic aneurysm repair once the proximal aorta is controlled, a patient who suffers a ruptured aneurysm with systemic hypotension requires timely and effective resuscitation, and our experience with out-of-hospital cardiopulmonary resuscitation by trained paramedics[57] suggests that significant salvage of life may be effected by such intervention. The use of the pneumatic antishock garment as a nonpharmacologic vasoconstrictor appears to have real merit in patients with ruptured aneurysms[58]; we have successfully air-evacuated such patients up to 800 miles in this fashion.

Establishment of proximal aortic control is all-important in patients with rupture of an abdominal aortic aneurysm: this is generally obtained by clamping or compressing the infradiaphragmatic aorta through a long midline incision. Some have advocated aortic control by clamping the distal thoracic aorta through a left thoracotomy,[59] whereas Sensenig[60] has reported a technique of emergency intraluminal occlusion of the abdominal aorta by a large Fogarty catheter passed retrograde from the left brachial artery into the thoracic aorta, then carried downstream under fluoroscopic control to a proper infrarenal position.

A number of interesting and vexing lesions may present concurrently with an abdominal aortic aneurysm, thus complicating surgical management. Hardy and Timms give a good summary of several of these.[61] For example, an unusual simultaneous occurrence of horseshoe kidney and abdominal aortic aneurysm may occur,[62] potentially leading to technical problems involving preservation of renal blood flow while doing aneurysmorrhaphy. Erosion of an aortic aneurysm into nearby viscera, including the inferior vena cava[63] or the intestinal tract,[64] may occur, with a patient showing massive congestive heart failure or exsanguinating gastrointestinal hemorrhage. Development of an "inflammatory" variant of abdominal aortic aneurysm has been mentioned previously; its cause remains obscure, but the technical consequences are very clear, including intense scarring and involvement of surrounding tissues with resulting jeopardy to these structures during operative dissection. Bacterial infection of an aortic aneurysm can lead to rupture or generalized septicemia (or both),[17] and because placement of a prosthetic graft into an infected area is interdicted, aortic ligature and extra-anatomic grafting—usually axillofemoral bypass —constitute the accepted operative procedure for these patients.[65] When such aneurysms are unruptured and can be resected en bloc, we have been successful with radical aortic excision and in situ graft interposition.[66] The coexistence of other intra-abdominal disease—colon cancer, for example, or cholelithiasis—is not uncommon, and the proper timing of management, as well as the wisdom of combining aneurysm resection with operative treatment of the second condition, remains unsettled.[67]

Rarely, a patient with a very large or symptomatic aneurysm may be in too precarious a medical condition to undergo transperitoneal aneurysmorrhaphy. In such patients, Leather and co-workers[68] and Berguer and associates[69] have promoted the use of extra-anatomic bypass grafting combined with iliac artery ligature, sometimes adding angiographic transcatheter coagulation of the aneurysm using thrombotic agents or surgical glue. In these workers' hands this technique has been quite successful, though distal ligature of an aneu-

rysm may occasionally be followed by aneurysmal rupture because of increased intraluminal pressure.

Outcome

Szilagyi and colleagues[29] have shown that surgical repair of an abdominal aortic aneurysm at least doubles the survival time a patient could expect should the aneurysm remain untreated. They also noted that, in untreated patients, the most common cause of death was rupture of the aneurysm, echoing the results reported by Estes.[41] The benefits of the operation obviously do not diminish the risk of cardiac death, and myocardial infarction remains the commonest cause of subsequent demise in patients who undergo successful repair of an abdominal aortic aneurysm.[29,31] This has led, in some centers, to an aggressive search for operable coronary artery lesions in patients harboring an aneurysm.[70] DeBakey and co-workers have also demonstrated the contribution of aneurysmorrhaphy to the prolongation of life.[71]

Successful repair of an abdominal aortic aneurysm thus appears to return patients toward the normal age-matched actuarial survival curve. Operative mortality for elective aneurysmorrhaphy should be well under 5%, and in skilled hands operative mortality rates of as low as 1% to 2% are common, despite the advanced age of patients who have aneurysm. Patients undergoing urgent operation because of symptomatic aneurysm should be expected to survive at almost the same rate. Those persons whose aneurysms rupture, and whose clinical course is characterized by significant hypotension, appear to have *at best* a 50% survival—much less in circumstances in which expert prehospital resuscitation and experienced vascular surgical skills are not available.

Summary

Abdominal aortic aneurysms are common, lethal lesions that appear to arise on the basis of biochemical or structural deficiencies; these may in part be hereditary. Aneurysmal degeneration appears to be a systemic disease affecting large and medium-sized arteries, clinically significant in the infrarenal aorta in the sixth, seventh and eighth decades of life. Elective operative repair, in skilled hands, carries less than a 5% operative mortality rate, even in an octogenarian population. On the other hand, survival following operation for aneurysmal rupture is much less than 50% at most institutions. No nonoperative management has been found to arrest the inexorable progression of these lesions. Thus, because the presence of an aortic aneurysm is generally the greatest risk to a patient's survival, early elective operation is warranted in all persons who are acceptable operative candidates.

REFERENCES

1. Blakemore AH: Progressive constrictive occlusion of the abdominal aorta with wiring and electrothermic coagulation. Ann Surg 1951; 133: 447-451

2. Creech O: Endoaneurysmorrhaphy and treatment of aortic aneurysm. Ann Surg 1966; 164:435-440

3. Osler W: Aneurysm of the abdominal aorta. Lancet 1905; 2:189-190

4. Carlsson J, Stendy NH: Aortic aneurysms. Acta Chir Scand 1964; 127:466-470

5. Rutherford RB: Infrarenal aortic aneurysms, *In* Rutherford RB (Ed): Vascular Surgery. Philadelphia, WB Saunders, 1977, pp 639-654

6. Crane C: Arteriosclerotic aneurysms of the abdominal aorta: Some pathological and clinical correlates. N Engl J Med 1955; 253:954-957

7. Gosling RG, Newman DL, Bowden NLR, et al: The area ratio of normal aortic junctions—Aortoiliac configuration and pulse-wave reflection. Br J Radiol 1971 Nov; 44:850-853

8. Roach MR: An experimental study of the production and time course of post-stenotic dilatation in the femoral and carotid arteries of adult dogs. Circ Res 1963; 13:537-542

9. Wolinsky H, Glagov S: Comparison of abdominal and thoracic aortic and medial structure in mammals—Deviation of man from the usual patterns. Circ Res 1969; 25:677-682

10. Tilson MD, Dang C: Generalized arteriomegaly: A possible predisposition to the formation of aneurysms. Arch Surg 1981; 116:1030-1032

11. Rowe DW, McGoodwin EB, Martin GR, et al: A sex-linked defect in the cross-linking of collagen and elastin associated with the mottled locus in mice. J Exp Med 1974; 139:180-185

12. Danks DM, Cartwright DE, Stevens BJ, et al: Menkes' kinky hair disease; further definition of the defect in copper transport. Science 1973; 179:1140-1142

13. Busuttil RW, Abou-Zamzam AM, Machleder HI: Collagenase activity of the human aorta: A comparison of patients with and without abdominal aortic aneurysms. Arch Surg 1980; 115:1373-1378

14. Dobrin PB: Mechanical properties of arteries. Physiol Rev 1978; 58:397-460

15. Swanson RJ, Littooy FW, Hunt TK, et al: Laparotomy as a precipitating factor in the rupture of abdominal aneurysms. Arch Surg 1980; 115:299-302

16. Tilson MD, Stansel HC: Differences in results for aneurysms versus occlusive disease after bifurcation grafts. Arch Surg 1980; 115:1173-1178

17. Penn I: Tuberculous aneurysm of the abdominal aorta. Br J Surg 1962; 50:288-292

18. Whelan TJ, Baugh JH: Nonatherosclerotic arterial lesions and their management—II. Inflammatory lesions of arteries. Curr Prob Surg 1967 Feb, pp 46-76

19. Sower ND, Whelan TJ: Suppurative arteritis due to salmonella. Surgery 1962; 52:841-854

20. Murdoch JL, Walker BA, Halprin BL, et al: Life expectancy and causes of death in the Marfan syndrome. N Engl J Med 1972; 286:804-806

21. Hunter JA, DeLaria GA: Abdominal aortic surgery in thoracic dissection, *In* Bergan JJ, Yao J (Eds): Aneurysms—Diagnosis and Treatment. New York, Grune & Stratton, 1982, pp 97-103

22. Griffen WO, Belin RP, Arnold IW: Traumatic aneurysm of the abdominal aorta. Surgery 1966; 60:813-817

23. Johnson JM, Gaspar MR, Movius HJ, et al: Sudden complete thrombosis of aortic and iliac aneurysms. Arch Surg 1974; 108:792-795

24. Siebert WT, Natelson EA: Chronic consumption coagulopathy accompanying abdominal aortic aneurysm. Arch Surg 1976; 111:539-544

25. Darling RC, Messina CR, Brewster DC, et al: Autopsy study of unoperated abdominal aortic aneurysm. Circulation 1977; 56(Suppl 2): 161-170

26. Kouchoukos NT, Levy JF, Butcher HR: Mortality from ruptured aortic aneurysm. Am J Surg 1967; 113:232-236

27. Kadir S, Athanasoulis CA, Brewster DC, et al: Tender pulsatile abdominal mass: Abdominal aortic aneurysm or not? Arch Surg 1980; 115:631-633

28. DeBakey ME: Études cliniques, chap 5, *In* Dubost G, Guilmet D, Soyer R (Eds): La Chirurgie des aneurismes de l'aorte. Paris, Masson et Cie, 1970, p 38

29. Szilagyi DÉ, Smith RD, DeRusso FJ, et al: Contribution of abdominal aneurysmectomy to prolongation of life. Ann Surg 1966; 164: 678-684

30. Dent TL, Lindenauer SM, Ernst CB, et al: Multiple arteriosclerotic aneurysms. Arch Surg 1972; 105:339-344

31. Hertzer NR: Fatal myocardial infarction following abdominal aortic aneurysm resection: 343 patients followed 6-11 years postoperatively. Ann Surg 1980; 192:667-673

32. Brewster DC, Darling RC, Raines JK, et al: Assessment of abdominal aortic aneurysm size. Circulation 1977; 56(Suppl 2):164-168

33. Hertzer NH, Beven EG: Ultrasound measurement and elective aneurysmectomy. JAMA 1978; 240:1966-1968

34. Brewster DC, Retina A, Waltman AC, et al: Angiography and the management of aneurysms of the abdominal aorta: Its value and safety. N Engl J Med 1975; 292:822-825

35. Satiani B, Veazey CR, Smith RB, et al: Preoperative aortography before abdominal aortic aneurysmectomy? Am Surg 1978 Oct; 44:650-654

36. Nusbaum JW, Freimanis AK, Thomford NR: Echography in the diagnosis of abdominal aortic aneurysm. Arch Surg 1971 Apr; 102:385-388

37. Bernstein EF, Dilley RB, Goldberg LE, et al: Growth rates of small abdominal aortic aneurysms. Surgery 1976; 80:765-770

38. Raskin MM, Cunningham JB: Comparison of computed tomography and ultrasound for abdominal aortic aneurysm: A preliminary study. J Comput Tomogr 1978; 2:21-23

39. Garrett HE, Ilabaca PA: Ruptured abdominal aortic aneurysm, *In* Bergan JJ, Yao J (Eds): Aneurysms—Diagnosis and Treatment. New York, Grune & Stratton, 1982, pp 303-314

40. Shumacker HB Jr, Barnes DL, King H: Ruptured abdominal aortic aneurysms. Ann Surg 1973 Jun; 177:772-779

41. Estes E: Abdominal aortic aneurysm: A study of 102 cases. Circulation 1950; 29:258-265

42. Klippel AP, Butcher HR: The unoperated abdominal aortic aneurysm. Am J Surg 1966; 111:629-635

43. Bernstein EF: Ultrasound techniques in the diagnosis and evaluation of abdominal aortic aneurysms, *In* Bernstein EF (Ed): Noninvasive Diagnostic Techniques in Vascular Disease. St Louis, CV Mosby, 1978, pp 330-338

44. Wolf EA, Sumner DS, Strandness DE: The relationship between abdominal aortic aneurysm and occlusive arterial disease. Surgery 1971 Oct; 103:480-483

45. Bandyk DF, Florence HG, Johansen KH: Ischemia accompanying ruptured abdominal aortic aneurysms. J Surg Res 1981; 30:297-302

46. Darling RC: Ruptured atherosclerotic abdominal aortic aneurysms: A pathologic and clinical study. Am J Surg 1970; 119:397-450

47. O'Donnell TF, Darling RC, Linton RR: Is 80 years too old for aneurysmectomy? Arch Surg 1980; 111:1250-1256

48. Worthington MM: Progress in transfusion therapy in treatment of bleeding problems in aortic aneurysm surgery. World J Surg 1980; 4: 521-526

49. Saleh SA: Anesthesia and monitoring for aortic aneurysm surgery. World J Surg 1980; 4:689-692

50. Barker WF: Major Problems in Clinical Surgery—Vol 4, Peripheral Arterial Disease, 2nd Ed. Philadelphia, WB Saunders, 1975, pp 403-446

51. Darling RC, Brewster DC: Elective treatment of abdominal aortic aneurysms. World J Surg 1980; 4:661-667

52. Luft HS, Bunker JP, Enthoven AC: Should operations be regionalized? The empirical relation between surgical volume and mortality. N Engl J Med 1979; 301:1364-1369

53. Bush HL, Logerfo FW, Wiesel RE, et al: Assessment of myocardial performance and optimal volume loading during elective abdominal aortic aneurysm resection. Arch Surg 1977; 112:130-138

54. Thomas GI, Jones TW, Stavney LS, et al: Experiences with autotransfusion during abdominal aortic aneurysm resection. Am J Surg 1980; 139:628-632

55. Thompson JE, Hollier LH, Patman RD, et al: Surgical management of abdominal aortic aneurysm. Am J Surg 1975; 181:654-662

56. Tchirkow G, Beven EG: Leg ischemia following surgery for abdominal aortic aneurysm. Am J Surg 1978 Aug; 188:166-170

57. Cobb LA, Alvarez H, Copass MD: A rapid response system for out-of-hospital cardiac emergencies. Med Clin North Am 1976; 60:283-290

58. Burn N, Lewis DG, Mackenzie A, et al: The G-suit: Its use in emergency surgery for ruptured abdominal aortic aneurysm. Anaesthesia 1972; 27:423-428

59. Merendino KA: Ruptured abdominal aortic aneurysm—Transthoracic control. Surg Gynecol Obstet 1956; 103:639-642

60. Sensenig DM: Rapid control in ruptured abdominal aortic aneurysm. Arch Surg 1981; 116:1034-1036

61. Hardy JD, Timms HH: Abdominal aortic aneurysm: Special problems. Ann Surg 1971; 73:945-955

62. Connelly TL, McKinnon W, Smith RB, et al: Abdominal aortic surgery and horseshoe kidney: Report of six cases and a review. Arch Surg 1980; 115:1459-1465

63. Beall AC, Cooley DA, Morris GC, et al: Perforation of atherosclerotic aneurysms into inferior vena cava. Arch Surg 1963; 86:136-143

64. Reckless JP, McColl I, Taylor GW: Aorto-enteric fistulae: An uncommon complication of abdominal aortic aneurysm. Br J Surg 1972; 59:458-463

65. Scher LA, Brener BJ, Goldenkranz RJ, et al: Infected aneurysms of the abdominal aorta. Arch Surg 1980 Aug; 115:975-978

66. Johansen K, Devin J: Mycotic aortic aneurysms—A reappraisal. Arch Surg 1983; 118:583-588

67. Szilagyi DE, Elliot JP, Berguer R: Coincidental malignancy and abdominal aortic aneurysm. Arch Surg 1967; 95:403-407

68. Leather RP, Shah DM, Goldman R, et al: Nonresective treatment of abdominal aortic aneurysm: Use of acute thrombosis and axillofemoral bypass. Arch Surg 1979; 114:1402-1406

69. Berguer R, Feldman AJ, Karmody AM: Intravascular thrombosis of abdominal aortic aneurysm in high-risk patients. Vasc Diag Ther, 1979; 1:24-26

70. Hertzer NM: Myocardial ischemia. Surgery 1983; 93:97-101

71. DeBakey ME, Crawford ES, Cooley DA, et al: Aneurysm of abdominal aorta: Analysis of graft replacement therapy 1-11 years after operation. Ann Surg 1964; 160:622-630

Medical Practice Questions

EDITOR'S NOTE: *From time to time medical practice questions from organizations with a legitimate interest in the information are referred to the Scientific Board by the Quality Care Review Commission of the California Medical Association. The opinions offered are based on training, experience and literature reviewed by specialists. These opinions are, however, informational only and should not be interpreted as directives, instructions or policy statements.*

Liver Transplantation

QUESTION:

Is liver transplantation considered established medical practice or does it remain investigational?

OPINION:

In the opinion of the Scientific Advisory Panels on General Surgery and Internal Medicine, and the Committee on Organ and Tissue Transplantation, liver transplantation is considered accepted medical practice for patients carefully selected by experienced medical transplant teams in centers with the appropriate resources and commitment to support such a program.

Published reports have shown that cyclosporine is an effective immunosuppressive agent and that its use has significantly contributed to the improved survival of liver transplant patients. Since 1980 one-year survival rates have been reported as high as 70%; projections for longer term survival are promising. Individual patients have survived many years with good quality of life after transplantation.

Liver transplantation offers a reasonable therapeutic approach for some patients with end-stage liver disease who have exhausted other available medical and surgical treatment. Though liver transplantation merits broader application, as recommended by the National Institutes of Health Consensus Development Conference statement on liver transplantation, June 20-23, 1983, its full potential will be defined ultimately by continued research and coordinated evaluation. The National Institutes of Health report concludes, and this opinion concurs, that this evaluation "can best be achieved by expansion of this technology to a limited number of centers where performance of liver transplantation can be carried out under optimal conditions."